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Parental physical activity and risk of obesity in their adult offspring: The HUNT study, Norway

BEV 3901 Master Thesis

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Abstract

Background: The occurrence of adiposity has increased in recent decades, and the detrimental consequences of obesity on health and disease constitutes a major challenge for public health. Parental obesity is a strong determinant of offspring obesity. This is in part due to genetic influences, but also environmental factors are of importance. Physical activity is a consistent and robust indicator associated with good health and reduced mortality, and a significant familial influence has been found linked to exercise participation. An association between parents' health behaviors and body mass index (BMI) in offspring has also been reported. The aim of this study was to examine parental physical activity habits in relation to BMI and risk of obesity in their adult offspring.

Methods: The study was based on data from 18493 father-offspring pairs and 17885 mother-offspring pairs who attended at least one of the two latest waves of the Nord-Trøndelag Health Study (HUNT 2 and HUNT 3). Height and weight were measured, and participants completed a detailed questionnaire including questions on physical activity. Mean differences in offspring BMI were estimated between categories of parental physical activity using a general linear model, and logistic regression was used to calculate odds ratio (OR) for offspring obesity (BMI $\geq$30 kg/m^2) associated with parental physical activity.

Results: Offspring with physically active parents showed lower mean BMI and lower risk of obesity in adulthood than offspring with less active or inactive parents. A statistically significant dose-response -relation between parental level of physical activity and risk of obesity in offspring showed that the association strengthened with increasing parental physical activity level. Mothers had a slightly stronger association with offspring than fathers. In relation to fathers and mothers high level of physical activity, the adjusted OR (95 % confidence interval) for offspring obesity was 0.76 (0.66 to 0.89) and 0.64 (0.54 to 0.76) compared to physically inactive parents.

Conclusion: In this population-based family linkage study, we found an inverse association between parental physical activity and offspring BMI for all measures of physical activity. Offspring of parents who were physically active had a lower risk of obesity than offspring of parents who were less active or inactive, and this was shown by a significant dose-response relation. The mother-offspring association was slightly stronger than the father-offspring association.

Contents

Introduction

Adiposity has increased in recent decades, and is now one of the most serious threats to public health (Lobstein, Baur, & Uauy, 2004). Obesity increases the risk of serious and chronic diseases, such as diabetes, cardiovascular disease (CVD), and some cancers (Bergström, Pisani, Tenet, Wolk, & Adami, 2001; Isomaa et al., 2001; Koh-Banerjee et al., 2004). A review reported that numerous studies have shown that obese individuals have higher all-cause mortality (Katzmarzyk, Janssen, & Ardern, 2003). The World Health Organization report obesity as one of the largest current health problems in the world, leading to huge costs for both the individual and the society (WHO, 2000).

Overweight parents tend to have overweight children. It is well established that parental obesity is one of the strongest determinants of offspring obesity (Garn, Sullivan, & Hawthorne, 1989; Maffeis, Talamini, & Tato, 1998; Whitaker, Wright, Pepe, Seidel, & Dietz, 1997). Part of this association is due to genetic influence. A review by Maes, Neale, and Eaves (1997), for example, suggests that genetic factors play a significant role in the causes of individual differences in relative body weight and adiposity. Several adoption and twin studies also support genetic influence on body mass index (BMI) (Silventoinen, Rokholm, Kaprio, & Sorensen, 2010; Wardle, Carnell, Haworth, & Plomin, 2008). On the other hand, the rapid recent increase in worldwide obesity has drawn attention to environmental factors. Davison and Birch (2002) suggest that characteristics of the family environment explain differences in children's weight status beyond that explained by genetic susceptibility. They refer to similarities between family members in behavioral risk factors associated with being overweight, including energy and percent fat intake, food preferences and physical activity. Most likely, a combination of both genes and the environment cause individuals to become overweight and obese.

Physical activity is an important factor in preventing obesity and a well-known indicator associated with good health and reduced mortality. Besides impacting positively on cardiovascular diseases, physical activity has also shown favorable effects on mental health, musculoskeletal disorders, and cancer (Batty & Thune, 2000; Dunn, Trivedi, Kampert, Clark, & Chambliss, 2005; Suominen, 2006). Despite this, research indicates that among worldwide populations people are less physically active and less physically fit than ever before, and that the average weight of children, adolescents, and adults has increased both in Norway and

internationally (Midthjell et al., 1999; WHO, 2000). Studies of trans-generational parent-offspring associations show a significant familial influence on leisure-time exercise participation (Beunen & Thomis, 1999; Simonen et al., 2002). Simonen et al. (2002) investigated a total of 200 families with information about physical activity in the Québec family study. Their findings suggest the presence of significant familial resemblance in physical activity and inactivity levels. Moreover, results from twin studies have shown that transgenerational associations reflect shared genes of family members (Lauderdale et al., 1997; Maia, Thomis, & Beunen, 2002). Stubbe et al. (2006) conducted a study on 37051 twin pairs from seven different countries. They found genetic factors contributed significantly to leisure-time exercise participation, and points out personality, proved to be highly heritable, as an important factor.

The majority of research so far has focused, firstly, on how overweight and obesity is related among family members, and secondly, on how health behavior, like activity habits, is transferred between generations. As far as we know, just a few studies have examined whether parental physical activity level and physical fitness are related to risk of offspring being overweight and obese. Burke, Beilin, and Dunbar (2001), examined longitudinal data from a cohort of Western Australian children and found that health behaviors and BMI aggregate within families, and an association between parents' health behaviors and BMI in offspring. A study by Fasting, Nilsen, Holmen, and Vik (2011) of adolescents from the same study population as the present study found that healthy changes in parental life-style, such as remaining normal weight and quitting smoking, were associated with lower occurrence of offspring adolescent overweight, but no clear association with parental physical activity was observed.

The aim of the current study was to use a large family-linkage database to examine if parental physical activity is associated with BMI and obesity in their adult offspring.

Materials and methods

Study population

The Nord-Trøndelag Health Study (HUNT) is a population-based health survey conducted in the Nord-Trøndelag County, in mid-Norway. To date, three cross-sectional waves have been completed; HUNT 1 (1984-86), HUNT 2 (1995-97) and HUNT 3 (2006-08). The population in Nord-Trøndelag County is stable and homogenous, making it suitable for epidemiological studies (Krokstad et al., 2012). It is also fairly representative of Norway in terms of geography, economy, and industry, source of income, morbidity and mortality (Holmen et al., 2003). Every citizen aged 20 or older has been invited to participate in the study and information has been collected through questionnaires, interviews, clinical measurements and biological samples from 125 000 participants. In HUNT 1, 86404 residents were invited to participate and 87 % (n = 77212) accepted; in HUNT 2, 93898 were invited and 70 % (n = 65237) accepted; and in HUNT 3, 93860 were invited and 54 % (n = 50807) accepted. Information about a wide range of health and lifestyle related factors was obtained through questionnaires, whereas physiological and anthropometric measurements were obtained at a clinical examination by trained personnel using standardized procedures (Holmen et al., 2003). A more detailed description of the HUNT study can be found at www.hunt.ntnu.no (accessed May 2013).

The present study is a family-linkage study based on data from participants in the two latest waves of the HUNT study; HUNT 2 and HUNT 3. Offspring have been linked to their biological parents through a national Family Register using their personal identification number. In total, the study sample consist of 18493 father-offspring pairs and 17885 mother-offspring pairs, constituting 17346 complete father-mother-offspring trios with sufficient information on physical activity and body mass index.

Study variables

BMI

Height (to the nearest 1.0 cm) and weight (to the nearest 0.5 kg) were measured with participants wearing light clothes without shoes. BMI was calculated as bodyweight in kilograms divided by the squared value of body height in meters (kg/m^2). BMI values were recoded into a categorical score and classified into four levels; underweight (<18.5 kg/m^2), normal weight (18.5-24.9 kg/m^2), overweight (25.0-29.9 kg/m^2) and obese (≥ 30.0 kg/m^2).

For the purpose of analyzing the association between parental physical activity and offspring obesity, offspring BMI was dichotomized into two categories; < 30 kg/m^2 and ≥ 30 kg/m^2 (obese).

Physical activity

In both HUNT 2 and HUNT 3 participants answered questions about leisure time physical activity habits. The questions involved how many hours of "light" and "hard" physical activity the participants conducted per week in the year prior to completing the questionnaire. "Hard" physical activity was described as getting sweaty or breathless, while "Light" physical activity was described as not getting sweaty or breathless. The participants had four possible response options for both questions; "none", "< 1 hour", "1-2 hours", or "≥ 3 hours". The answers to these two questions were recoded into an index variable and classified into four different groups; "no activity" defined as not doing any exercise (inactive); "low" defined as < 1 hour light and no hard activity; "medium" defined as ≥ 1 hour light and/or < 1 hour hard activity; and "high" defined as any light and ≥ 1 hour hard activity.

Other factors

Smoking status

The participants were asked about their smoking history and smoking habits, such as past and present daily smoking, number of cigarettes, time since quitting smoking, etc. This information was used to classify participants into four categories of smoking; never, former, current smoker, or unknown.

Parental age

Considering the age difference between the parents and the offspring in this study, and that age probably correlate with both weight and level of physical activity, we decided to take this variable into account in the analysis.

Ethics

The study has been approved by the Regional Committee for Ethics in Medical Research. Participation in the study was voluntary and each participant signed a written consent upon participation.

Statistical analyses

Characteristics of the study population were analyzed using descriptive statistics and presented as frequencies and percentages, or as means with standard deviations (SD). The association between parental physical activity and offspring BMI and obesity was studied in two different models. First, we used a general linear model to estimate the adjusted mean difference in offspring BMI between categories of parental physical activity, and next, we used logistic regression to calculate odds ratio (OR) as a measure of relative risk for offspring obesity associated with parental physical activity. The precision of the estimated association was assessed by a 95 % confidence interval (CI). We also conducted a trend test across categories of physical activity by entering the categories as an ordinal variable in the regression model. All associations were adjusted for confounding by parental age (continuous), parental smoking status (never, former, current, unknown), and parental BMI (underweight, normal weight, overweight, obese). Finally, to examine if the parent-offspring association was modified by offspring age, we repeated the logistic regression analyses stratified by the median offspring age (37.6 years).

All statistical analyses were conducted using SPSS 20.0 for Windows (© SPSS Inc., 1989-2011).

Results

Characteristics of the study population

Characteristics of parents and offspring are presented in Table 1. The number of offspring included in this table is slightly fewer than those included in the main analyses, since information on physical activity had to be available. Mean age of parents was 60.1 years in fathers and 56.7 years in mothers. Mean offspring age was 38.3 years in sons and 37.5 years in daughters. Based on BMI, 35 % of mothers and 28 % of fathers were classified as normal weight, whereas 50 % of daughters and 35 % of sons were normal weight. Correspondingly, 41 % of mothers and 54 % of fathers were overweight, compared to 32 % of daughters and 48 % of sons. Finally, 24 % of mothers, 18 % of fathers, 16 % of daughters and 17 % of sons were obese. Regarding the physical activity level, 8 % of fathers and 9 % mothers were inactive, compared to 4 % of sons and 2 % of daughters. A total of 46 % of sons and 45 % of daughters reported a high activity level (at least 1 hour or more of hard physical activity per week), in contrast to 27 % of fathers and 17 % of mothers.

Table 1: Descriptive statistics.

	Fathers	Mothers	Sons	Daughters
Number of participants	18493	17885	8154	9192
Age, mean years (SD)	60.1 (11.9)	56.7 (11.8)	38.3 (11.7)	37.5 (11.6)
BMI, mean kg/m^2 (SD)	27.1 (3.5)	27.2 (4.7)	26.7 (3.8)	25.8 (4.7)
Physical Activity				
No activity, n (%)	1460 (7.9)	1560 (8.7)	355 (4.4)	184 (2.0)
Low, n (%)	5446 (29.4)	7257 (40.6)	1548 (19.0)	1864 (20.3)
Medium, n (%)	6561 (35.5)	6004 (33.6)	2511 (30.8)	3053 (33.2)
High, n (%)	5026 (27.2)	3064 (17.1)	3740 (45.9)	4091 (44.5)
BMI, kg/m^2				
Underweight, n (%)	42 (0.2)	115 (0.6)	37 (0.5)	115 (1.3)
Normal weight, n (%)	5150 (27.8)	6217 (34.8)	2818 (34.6)	4593 (50.0)
Overweight, n (%)	9928 (53.7)	7283 (40.7)	3937 (48.3)	2975 (32.4)
Obese, n (%)	3373 (18.2)	4249 (23.8)	1362 (16.7)	1507 (16.4)

N = number of participants, BMI = body mass index, SD = standard deviation.
Underweight BMI = < 18.50, Normal weight BMI = 18.50-24.99, Overweight BMI = 25-29.99, Obese BMI = ≥ 30. In terms of physical activity, **no activity** was defined as not doing any exercise (inactive), **low** was defined as < 1 hour light and no hard activity, **medium** was defined as 1 hour light and/or < 1 hour hard activity, and **high** was defined as any light and ≥ 1 hour's hard activity.

Table 2: Parental physical activity in relation to offspring mean body mass index (BMI, kg/m^2).

		N	Mean offspring BMI	Crude diff.	Adj. diff[a]	95% CI
Fathers						
Light	No activity	1506	26.73	0.0	0.0	Reference
	<1 h. per week	2931	26.51	- 0.22	- 0.08	(- 0.35 to 0.18)
	1-2 h. per week	5959	26.15	- 0.58	- 0.41	(- 0.65 to - 0.67)
	3+ h. per week	6382	26.14	- 0.59	- 0.40	(- 0.64 to - 0.16)
Hard	No activity	4972	26.68	0.0	0.0	Reference
	<1 h. per week	3458	25.92	- 0.76	- 0.54	(- 0.72 to - 0.35)
	1-2 h. per week	3006	25.83	- 0.85	- 0.53	(- 0.72 to - 0.34)
	3+ h. per week	2020	25.76	- 0.92	- 0.66	(- 0,88 to - 0.44)
Index	No activity	1460	26.76	0.0	0.0	Reference
	Low	5446	26.48	- 0.29	- 0.24	(- 0.48 to - 0.01)
	Medium	6561	26.16	- 0.61	- 0.44	(- 0.69 to - 0.20)
	High	5026	25.81	- 0.96	- 0.60	(- 0.85 to - 0.35)
Mothers						
Light	No activity	1536	27.08	0.0	0.0	Reference
	<1 h. per week	2955	26.39	- 0.69	- 0.36	(- 0.62 to - 0.09)
	1-2 h. per week	6860	26.13	- 0.96	- 0.46	(- 0.70 to - 0.22)
	3+ h. per week	5636	25.95	- 1.13	- 0.56	(- 0.80 to - 0.31)
Hard	No activity	5981	26.57	0.0	0.0	Reference
	<1 h. per week	2757	25.69	- 0.88	- 0.63	(- 0.82 to - 0.44)
	1-2 h. per week	2226	25.52	- 1.06	- 0.72	(- 0.93 to - 0.52)
	3+ h. per week	838	25.25	- 1.33	- 0.92	(- 1.23 to - 0.62)
Index	No activity	1560	27.05	0.0	0.0	Reference
	Low	7257	26.4	- 0.65	- 0.31	(- 0.55 to - 0.08)
	Medium	6004	25.95	- 1.10	- 0.60	(- 0.84 to - 0.36)
	High	3064	25.44	- 1.61	- 0.94	(- 1.20 to - 0.67)

[a] Adjusted for parental age (continuous), BMI (kg/m^2) and smoking (never, former, current or unknown). N= number of participants, BMI = body mass index, CI = confidence interval.
In terms of the physical activity index, **no activity** was defined as not doing any exercise (inactive), **low** was defined as <1 hour light and no hard activity, **medium** was defined as 1 hour light and/or <1 hour hard activity, and **high** was defined as any light and ≥1 hour's hard activity.

Parental physical activity and offspring BMI

The association between parental physical activity and offspring mean BMI is presented in Table 2. There was evidence of an inverse association between parental physical activity and offspring BMI for all measures of physical activity, although the association was somewhat stronger for hard than for light activity. Offspring with fathers who reported three hours or more of light physical activity had a BMI that was -0.40 kg/m^2 (95 % CI, -0.64 to -0.16) lower than offspring with fathers who reported no light activity. The corresponding association related to mother's level of light activity was -0.56 kg/m^2 (95 % CI, -0.80 to -0.31). Analyses of hard activity gave mean differences in offspring BMI of -0.66 kg/m^2 (95 % CI, -0.88 to -0.44) and -0.92 kg/m^2 (95 % CI, -1.23 to -0.62) comparing ≥3 hour's hard activity versus no hard activity in fathers and mothers, respectively. Similar associations were observed for total physical activity (Index), with mean differences of -0.60 (95 % CI, -0.85 to -0.35) and -0.94 (95% CI, -1.20 to -0.67) comparing extreme categories of fathers and mothers activity level. Overall, these associations were slightly stronger for mothers than fathers.

Table 3: Parental physical activity and offspring's risk of obesity (BMI ≥30 kg/m^2).

		No. of obese	Not obese	OR[a]	OR[b]	95% CI
Fathers						
Light	No activity	314	1191	1.0	1.0	Reference
	<1 h. per week	548	2383	0.87	0.92	(0.78 to 1.08)
	1-2 h. per week	1006	4952	0.77	0.85	(0.73 to 0.98)
	3+ h. per week	1026	5354	0.73	0.82	(0.71 to 0.94)
Hard	No activity	998	3973	1.0	1.0	Reference
	<1 h. per week	541	2917	0.74	0.79	(0.70 to 0.89)
	1-2 h. per week	448	2558	0.70	0.79	(0.70 to 0.90)
	3+ h. per week	285	1735	0.65	0.74	(0.64 to 0.85)
Index	No activity	306	1153	1.0	1.0	Reference
	Low	1002	4443	0.85	0.88	(0.76 to 1.02)
	Medium	1087	5472	0.75	0.82	(0.71 to 0.95)
	High	733	4293	0.64	0.76	(0.66 to 0.89)
Mothers						
Light	No activity	352	1184	1.0	1.0	Reference
	<1 h. per week	524	2430	0.73	0.82	(0.70 to 0.97)
	1-2 h. per week	1103	5755	0.65	0.80	(0.69 to 0.92)
	3+ h. per week	846	4789	0.59	0.78	(0.67 to 0.90)
Hard	No activity	1140	4840	1.0	1.0	Reference
	<1 h. per week	378	2377	0.68	0.74	(0.65 to 0.84)
	1-2 h. per week	280	1946	0.61	0.71	(0.61 to 0.82)
	3+ h. per week	96	741	0.55	0.67	(0.53 to 0.83)
Index	No activity	351	1207	1.0	1.0	Reference
	Low	1285	5971	0.74	0.86	(0.74 to 0.98)
	Medium	905	5097	0.61	0.76	(0.66 to 0.88)
	High	376	2687	0.48	0.64	(0.54 to 0.76)

[a] Unadjusted [b] Adjusted for parental age (continuous), BMI (kg/m^2) and smoking (never, current, former or unknown). CI = confidence interval, OR = odds ratio. In terms of the physical activity index, **no activity** was defined as not doing any exercise (inactive), **low** was defined as <1 hour light and no hard activity, **medium** was defined as 1 hour light and/or <1 hour hard activity, and **high** was defined as any light and ≥1 hour's hard activity.

Parental physical activity and risk of obesity in offspring

Table 3 shows ORs for offspring obesity (BMI ≥30 kg/m^2). A trend test showed a significant dose-response relation between parental level of physical activity and risk of obesity in offspring (all p-trends < 0.001).

The adjusted OR for offspring obesity was 0.82 (95 % CI, 0.71 to 0.94) if fathers reported ≥3 hours per week of light physical activity compared to no light activity. The corresponding OR associated with mothers' light activity was 0.78 (95 % CI, 0.67 to 0.90). If fathers reported ≥3 hours of hard physical activity per week, the adjusted OR for offspring obesity was 0.74 (95 % CI, 0.64 to 0.85) compared to no hard activity, whereas in relation to mothers' hard activity the OR was 0.67 (95 % CI, 0.53 to 0.83). In relation to total physical activity (Index), the adjusted OR for offspring obesity was 0.82 (95 % CI, 0.71 to 0.95) when fathers reported medium activity and 0.76 (95 % CI, 0.66 to 0.89) when they reported high activity levels. The corresponding association for mothers activity level was 0.76 (95 % CI, 0.66 to 0.88) and 0.64 (95 % CI, 0.54 to 0.76). Similar to the results for offspring mean BMI, these results suggests that maternal activity show a slightly stronger association with the risk of offspring obesity, than paternal activity.

Table 4: Odds ratio (OR) for offspring obesity (BMI ≥30 kg/m^2) associated with the combined paternal and maternal total physical activity.

	Maternal total activity			
Paternal total activity	Inactive	Low	Moderate	High
Inactive				
Cases/non-cases	79/190	112/409	42/253	30/110
OR (95% CI)	1.00 (Reference)	0.76 (0.53-1.08)	0.54 (0.35-0.84)	0.80 (0.48-1.33)
Low				
Cases/non-cases	88/307	430/1806	241/1260	84/501
OR (95% CI)	0.77 (0.53-1.11)	0.69 (0.51-0.93)	0.61 (0.45-0.84)	0.55 (0.38-0.79)
Medium				
Cases/non-cases	97/299	380/1857	344/1927	115/771
OR (95% CI)	0.84 (0.58-1.22)	0.64 (0.48-0.87)	0.60 (0.44-0.81)	0.53 (0.38-0.75)
High				
Cases/non-cases	35/192	239/1310	224/1269	123/1110
OR (95% CI)	0.50 (0.31-0.79)	0.60 (0.44-0.82)	0.62 (0.46-0.86)	0.45 (0.32-0.64)

CI = confidence intervals, OR = odds ratio. In terms of the physical activity index, **no activity** was defined as not doing any exercise (inactive), **low** was defined as <1 hour light and no hard activity, **medium** was defined as 1 hour light and/or <1 hour hard activity, and **high** was defined as any light and ≥1 hour's hard activity.

Table 4 shows the ORs for offspring obesity when combining paternal and maternal total physical activity. The risk of offspring obesity decreased steadily when both paternal and maternal total activity changes from 'inactive' to 'high' physical activity. Relative to the first category (inactive mother and father), the OR was 0.69 (95 % CI, 0.51-0.93), when both reported low activity levels, 0.60 (95 % CI, 0.44 to 0.81) when they reported medium activity, and 0.45 (95 % CI, 0.32-0.64) when both parents reported high activity levels.

Discussion

Main findings

In this population-based family-linkage study in Norway, we found that parental physical activity level was inversely associated with offspring BMI. Offspring of parents who were physically active had lower mean BMIs and lower risk of obesity than those of parents who were less active or inactive. These associations became stronger the more physically active the parents were, as shown by a statistically significant dose-response -relation between parental level of physical activity and risk of obesity in offspring. We also found that the mother-offspring association was slightly stronger than the father-offspring association. Finally, the lowest risk of offspring obesity was observed when both parents reported a high physical activity level.

Strength and limitations

The study has several important strengths, including data collected in a large and representative sample from a population with wide age range (Krokstad et al., 2012). Together with a high participation rate and a thorough work with the questionnaires and clinical examinations, this reduces the possibility that chance findings and selection bias affect the results. The fact that the data are linked to the National Family Registry makes it possible to study family members across different generations.

HUNT covers an extensive range of topics. Hence, the data are diverse, allowing us to adjust for potentially confounding effects of factors like smoking and BMI. However, there will always be a possibility for residual confounding due to misclassification and unknown or unmeasured factors. For example, we did not have information on dietary habits of parents or offspring. Diet is known to be an important predictor of adiposity in the offspring, and this might have influenced our results (Gallant et al., 2013; Nguyen, Larson, Johnson, & Goran, 1996). Neither did we have information on parental socio-economic position, such as income or education. Socio-economic differences in physical activity and obesity have been reported (Giles-Corti & Donovan, 2002; Stamatakis, Primatesta, Chinn, Rona, & Falascheti, 2005), and could thus have biased our results.

Self-reported information collected in questionnaires can cause information bias and misclassification that could have influenced our results. However, height and weight were

measured objectively by trained personnel using standardized measurements (Krokstad et al., 2012), thus avoiding biases that are often affiliated with self-reported anthropometric data (Rowland, 1990). Information on physical activity was obtained from questionnaires that have been validated against objective measures of physical activity, energy expenditure, and cardiorespiratory fitness in a random sample of young men (Kurtze, Rangul, & Hustvedt, 2008). This validation found that the questionnaire was a moderately good measure of vigorous activity, and correlated well with measured oxygen consumption. However, the narrow sample of only young men included in this validation study may reduce the ability to generalize to the total sample included in the HUNT studies. Physical activity is a complex behavior, so misclassification cannot be excluded. Still, it has been reported that questionnaire are useful to classify people into broad categories of activity, such as low, medium and high activity (Shephard, 2003).

Comparison with existing literature

Several studies show that obesity aggregates within families as a result of interaction between genetic and environmental factors (Burke et al., 2001; Maffeis et al., 1998; Whitaker et al., 1997).

Previous studies have reported familial connections between weight status and influence on leisure time physical activity. Also, it seems a clear pattern exists in certain families who demonstrate a combination of dietary and activity habits that promote obesity development among family members beyond that explained by genetic susceptibility (Burke et al., 2001; Davison & Birch, 2002; Simonen et al., 2002). Twin studies have shown that environmental factors, such as lack of physical activity or overfeeding, are not sufficient to cause overweight. These suggest that genetic factors also play an important role (Rossow & Rise, 1994; Stubbe et al., 2006). The main perceptions seem to be that becoming overweight is most likely when a susceptible individual is situated in an adverse environment (Fasting et al., 2011).

To our knowledge, no previous study have examined the association between parental physical activity and their adult offspring BMI. A study similar to the present examined longitudinal data from a cohort of Australian children from the age of 9 to 18, investigating BMI and lifestyle factors such as diet and smoking habits (Burke et al., 2001). Their results

showed an association between health behaviours in parents and BMI in offspring, but parental physical activity was not measured. A study by Davison and Birch (2002) included 197 girls, measured at the age of 5 and then 7 years old, and their parents. The study aimed at identifying obesogenic families based on mothers' and fathers' dietary and activity patterns. The measures of parents' physical activity and dietary intake were entered into a cluster analysis. Obesogenic and non-obesogenic family clusters were identified. Mothers and fathers in the obesogenic cluster reported higher dietary intake scores and lower physical activity scores than mothers and fathers in the non-obesogenic cluster. This obesogenic pattern predicted differences in girls' BMI and skinfold thickness, even after controlling for parents' BMI. These findings are in line with our results. Furthermore, in a Norwegian study in parents from HUNT-1 (1984-86) to HUNT-2 (1995-97), researchers found that healthy life-style changes, such as weight loss and smoking cessation, were associated with lower occurrence of overweight in their adolescent offspring (Fasting et al., 2011). Results from the study also suggest that parental weight gain during their offspring's childhood constitutes an important risk factor for offspring becoming overweight later in life. Fasting et al. (2011), in contrast to our study, did not find any direct associations between level of parental physical activity and offspring becoming overweight in adolescence.

In the present study, we found that mother-offspring associations was slightly stronger than father-offspring associations. Fasting et al. (2011) also reported that the association was stronger for mothers than fathers regarding change in weight status and smoking habits. On the other hand, Rossow and Rise (1994) found that all parental health behaviours were significantly associated with that of their adolescent child, except for mother's exercise.

Possible mechanisms

The inverse relationship between physical activity and body fat or weight is well established, and confirmed in several studies (Ball, Owen, Salmon, Bauman, & Gore, 2001; Hemmingsson & Ekelund, 2007; Tremblay et al., 1990). Thus, physical activity plays an important role in preventing the development of overweight and obesity. However, it is clear that other factors also are essential in relation to body weight and body composition.

The strong association between parental and offspring overweight supports the theory of an inherited susceptibility to obesity (Burke et al., 2001; Maes et al., 1997). This association is

strongest if both parents are obese (Whitaker et al., 1997). Weight and height are both subject to genetic influence, confirmed by a number of studies (Maes et al., 1997; Schousboe et al., 2003; Weedon et al., 2008). Further, differences in associations between BMI and physical activity in obese and non-obese people have been found. These indicate that obesity may act as a barrier to physical activity (Hemmingsson & Ekelund, 2007). It has also been reported that genes may influence participation in physical activity. In a review by Bouchard, Malina, and Pérusse (1997), the studies being examined gave clear evidence that somatic dimensions, body composition and body type, known to be important factors in physical performance, are all under moderate to high genetic control. A genetic influence has also been reported for daily physical activity (Beunen & Thomis, 1999). This may help explain why some people are more likely to participate in physical activity than others, and serve as a possible context for the association between parental physical activity level and offspring BMI observed in this study. Furthermore, genes may influence regular participation in specific exercise more than moderate activity, such as walking (Beunen & Thomis, 1999). This could explain that the association is stronger for hard activity level than light activity level in our results. Simonen et al. (2002) found in their study that the highest heritability level was for the inactivity phenotype.

At the same time, there is strong evidence that environmental factors also contribute in determining both human adiposity and physical activity level (Maes et al., 1997; Simonen et al., 2002). Rossow and Rise's (1994) results suggest that health behaviors (in forms of smoking, alcohol consumption, fat intake and exercise) among parents and their adolescent offspring are strongly associated. One explanation given for the similarities between parents and offspring is that parents are more likely to purchase the food, plan and provide equipment for sports and leisure time activities. They are also more likely to set family rules and schedules that reflect their socioeconomic status and serve as social antecedents of health behaviors (Rossow & Rise, 1994). Thus, parent's behaviours related to food preference, tobacco use or physical activity might provide social cues for their children's behavior. When children reach adolescence and start the process of separating from their parents, they bring parental attitudes with them. The family resemblance in physical activity levels has also been found to persist beyond childhood and adolescent years, and after offspring move away from the family home (Rossow & Rise, 1994; Simonen et al., 2002). This may contribute to explaining the association between parents and the adult offspring in our study.

Aging is associated with increased body weight, while changes in total and regional fat distribution over an adult's lifespan may influence BMI (Kuk, Saunders, Davidson, & Ross, 2009). Considering the parents impact on offspring lifestyle, we expected that the association between parent and offspring would be strongest when the sons and daughters were younger and in the initial phase of establishing their own lives, and vaguer when they got older and more independent. To examine if this influenced our results, we stratified the analyses by the median offspring age (37.6 years). There was no differences in the associations for the two groups, and this suggests that the parent-offspring associations are robust even when the offspring are older adults.

As mentioned, in the present study the association between mothers and offspring was slightly stronger than between fathers and offspring. Several studies have investigated the possibility that maternal obesity during pregnancy led to an intrauterine environment that stimulates increased obesity among their offspring (Davey Smith, Steer, Leary, & Ness, 2007; Fleten et al., 2012; Lawlor et al., 2007). In an Australian cohort, they found a stronger association between maternal-offspring BMI than between paternal-offspring BMI, suggesting a potential role of the intrauterine environment (Lawlor et al., 2007). However, Fleten et al. (2012) found an association between maternal-offspring BMI and paternal-offspring BMI when the offspring was 3 years old. This indicates that the association is likely to be explained by shared familial risk rather than the intrauterine environment. This is supported by Davey Smith et al. (2007). Another explanation for stronger maternal than paternal associations could be the influence of non-paternity, when the registered father is not the biological father (Davey Smith et al., 2007; Macintyre & Sooman, 1991). Various rates of non-paternity have been reported, but no reliable estimate exists for the current population. However, an ongoing study of transgenerational associations of cardiovascular risk factors within the HUNT study suggests no large effects of non-paternity in these data (personal communication, Kirsti Lund Vik, Department of Human Movement Science, NTNU).

Conclusion

In this population-based family linkage study, parental physical activity level was inversely associated with BMI and obesity in their adult children. Offspring with physically active parents had lower mean BMIs and lower risk of obesity in adulthood than offspring with less active or inactive parents, and this was shown by a dose-response association. The mother-offspring association was slightly stronger than the father-offspring association. These results suggest that in future work towards overweight and obesity, family relations should be considered. The finding of parent-offspring associations in adult children may, furthermore, indicate that these associations last for a lifetime.

References

Ball, K., Owen, N., Salmon, J., Bauman, A., & Gore, C. J. (2001). Associations of physical activity with body weight and fat in men and women. *International Journal of Obesity and Related Metabolic Disorders, 25*(6), 914-919.

Batty, D., & Thune, I. (2000). Does physical activity prevent cancer? Evidence suggests protection against colon cancer and probably breast cancer. *British Medical Journal, 321*(7274), 1424-1425.

Bergström, A., Pisani, P., Tenet, V., Wolk, A., & Adami, H. O. (2001). Overweight as an avoidable cause of cancer in Europe. *International Journal of Cancer, 91*(3), 421-430.

Beunen, G., & Thomis, M. (1999). Genetic determinants of sports participation and daily physical activity. *International Journal of Obesity and Related Metabolic Disorders, 23* (3), 55-63.

Bouchard, C., Malina, R. M., & Pérusse, L. (1997). *Genetics of Fitness and Physical Performance*: Human Kinetics 1.

Burke, V., Beilin, L. J., & Dunbar, D. (2001). Family lifestyle and parental body mass index as predictors of body mass index in Australian children: a longitudinal study. *International Journal of Obesity and Related Metabolic Disorders, 25*(2), 147-157.

Davey Smith, G., Steer, C., Leary, S., & Ness, A. (2007). Is there an intrauterine influence on obesity? Evidence from parent–child associations in the Avon Longitudinal Study of Parents and Children (ALSPAC). *Archives of Disease in Childhood, 92*(10), 876-880.

Davison, K. K., & Birch, L. L. (2002). Obesigenic families: parents' physical activity and dietary intake patterns predict girls' risk of overweight. *International Journal of Obesity and Related Metabolic Disorders, 26*(9), 1186-1193.

Dunn, A. L., Trivedi, M. H., Kampert, J. B., Clark, C. G., & Chambliss, H. O. (2005). Exercise treatment for depression: efficacy and dose response. *American Journal of Preventive Medicine, 28*(1), 1-8.

Fasting, M. H., Nilsen, T. I., Holmen, T. L., & Vik, T. (2011). Changes in parental weight and smoking habits and offspring adiposity: data from the HUNT-study. *International Journal of Pediatric Obesity, 6*(2-2), 399-407.

Fleten, C., Nystad, W., Stigum, H., Skjærven, R., Lawlor, D. A., Davey Smith, G., & Næss, Ø. (2012). Parent-Offspring Body Mass Index Associations in the Norwegian Mother and Child Cohort Study: A Family-based Approach to Studying the Role of the Intrauterine Environment in Childhood Adiposity. *American Journal of Epidemiology, 176*(2), 83-92.

Gallant, A. R., Tremblay, A., Perusse, L., Despres, J. P., Bouchard, C., & Drapeau, V. (2013). Parental eating behavior traits are related to offspring BMI in the Quebec Family Study. *International Journal of Obesity (Lond).*

Garn, S .M., Sullivan, T. V., & Hawthorne, V. M. (1989). Fatness and obesity of the parents of obese individuals. *The American Journal of Clinical Nutrition, 50*(6), 1308-1313.

Giles-Corti, B., & Donovan, R. J. (2002). Socioeconomic Status Differences in Recreational Physical Activity Levels and Real and Perceived Access to a Supportive Physical Environment. *Preventive Medicine, 35*(6), 601-611.

Hemmingsson, E., & Ekelund, U. (2007). Is the association between physical activity and body mass index obesity dependent? *International Journal of Obesity (Lond), 31*(4), 663-668.

Holmen, J., Midthjell, K., Krüger, Ø., Langhammer, A., Holmen, T. L., Bratberg, G. H., . . . Larsen, P. G. L. (2003). The Nord-Trøndelag Health Study 1995-97 (HUNT 2): Objectives, contents, methods and participation. *Norsk Epidemiologi 13*(1), 19-32.

Isomaa, B., Almgren, P., Tuomi, T., Forsen, B., Lahti, K., Nissen, M., . . . Groop, L. (2001). Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes Care, 24*(4), 683-689.

Katzmarzyk, P. T., Janssen, I., & Ardern, C. I. (2003). Physical inactivity, excess adiposity and premature mortality. *Obesity Reviews, 4*(4), 257-290.

Koh-Banerjee, P., Wang, Y., Hu, F. B., Spiegelman, D., Willett, W. C., & Rimm, E. B. (2004). Changes in body weight and body fat distribution as risk factors for clinical diabetes in US men. *American Journal of Epidemiology, 159*(12), 1150-1159.

Krokstad, S., Langhammer, A., Hveem, K., Holmen, T.L., Midthjell, K., Stene, T.R., . . . Holmen, J. (2012). Cohort Profile: The HUNT Study, Norway. *International Journal of Epidemiology*.

Kuk, J. L., Saunders, T. J., Davidson, L. E., & Ross, R. (2009). Age-related changes in total and regional fat distribution. *Ageing Research Reviews, 8*(4), 339-348.

Kurtze, N., Rangul, V., & Hustvedt, B. E. (2008). Reliability and validity of the international physical activity questionnaire in the Nord-Trondelag health study (HUNT) population of men. *BMC Medical Research Methodology, 8*(1), 63.

Lauderdale, D. S., Fabsitz, R., Meyer, J. M., Sholinsky, P., Ramakrishnan, V., & Goldberg, J. (1997). Familial determinants of moderate and intense physical activity: a twin study. *Medicine and Science in Sports and Exercise, 29*(8), 1062-1068.

Lawlor, D. A., Smith, G. D., O'Callaghan, M., Alati, R., Mamun, A. A., Williams, G. M., & Najman, J. M. (2007). Epidemiologic Evidence for the Fetal Overnutrition Hypothesis: Findings from the Mater-University Study of Pregnancy and Its Outcomes. *American Journal of Epidemiology, 165*(4), 418-424.

Lobstein, T., Baur, L., & Uauy, R. (2004). Obesity in children and young people: a crisis in public health. *Obesity Reviews, 5 Suppl 1*, 4-104.

Macintyre, S., & Sooman, A. (1991). Non-paternity and prenatal genetic screening. *The Lancet, 338*(8771), 869-871.

Maes, H. H., Neale, M. C., & Eaves, L. J. (1997). Genetic and environmental factors in relative body weight and human adiposity. *Behavior Genetics, 27*(4), 325-351.

Maffeis, C., Talamini, G., & Tato, L. (1998). Influence of diet, physical activity and parents' obesity on children's adiposity: a four-year longitudinal study. *International Journal of Obesity and Related Metabolic Disorders, 22*(8), 758-764.

Maia, J. A., Thomis, M., & Beunen, G. (2002). Genetic factors in physical activity levels: a twin study. *American Journal of Preventive Medicine, 23*(2 Suppl), 87-91.

Midthjell, K., Kruger, O., Holmen, J., Tverdal, A., Claudi, T., Bjorndal, A., & Magnus, P. (1999). Rapid changes in the prevalence of obesity and known diabetes in an adult Norwegian population. The Nord-Trondelag Health Surveys: 1984-1986 and 1995-1997. *Diabetes Care, 22*(11), 1813-1820.

Nguyen, V. T., Larson, D. E., Johnson, R. K., & Goran, M. I. (1996). Fat intake and adiposity in children of lean and obese parents. *The American Journal of Clinical Nutrition, 63*(4), 507-513.

Rossow, I., & Rise, J. (1994). Concordance of parental and adolescent health behaviors. *Social Science and Medicine, 38*(9), 1299-1305.

Rowland, M. L. (1990). Self-reported weight and height. *The American Journal of Clinical Nutrition, 52*(6), 1125-1133.

Schousboe, K., Willemsen, G., Kyvik, K. O., Mortensen, J., Boomsma, D. I., Cornes, B. K., . . . Harris, J. R. (2003). Sex differences in heritability of BMI: a comparative study of results from twin studies in eight countries. *Twin Research, 6*(5), 409-421.

Shephard, R J. (2003). Limits to the measurement of habitual physical activity by questionnaires. *British Journal of Sports Medicine, 37*(3), 197-206.

Silventoinen, K., Rokholm, B., Kaprio, J., & Sorensen, T. I. (2010). The genetic and environmental influences on childhood obesity: a systematic review of twin and adoption studies. *International Journal of Obesity (Lond), 34*(1), 29-40.

Simonen, R. L., Perusse, L., Rankinen, T., Rice, T., Rao, D. C., & Bouchard, C. (2002). Familial aggregation of physical activity levels in the Quebec Family Study. *Medicine and Science in Sports and Exercise, 34*(7), 1137-1142.

Stamatakis, E., Primatesta, P., Chinn, S., Rona, R., & Falascheti, E. (2005). Overweight and obesity trends from 1974 to 2003 in English children: what is the role of socioeconomic factors? *Archives of Disease in Childhood, 90*(10), 999-1004.

Stubbe, J. H., Boomsma, D. I., Vink, J. M., Cornes, B. K., Martin, N. G., Skytthe, A., . . . de Geus, E. J. (2006). Genetic influences on exercise participation in 37,051 twin pairs from seven countries. *PLoS One, 1*, e22.

Suominen, H. (2006). Muscle training for bone strength. *Aging Clinical and Experimental Research, 18*(2), 85-93.

Tremblay, A., Després, J. P., Leblanc, C., Craig, C. L., Ferris, B., Stephens, T., & Bouchard, C. (1990). Effect of intensity of physical activity on body fatness and fat distribution. *The American Journal of Clinical Nutrition, 51*(2), 153-157.

Wardle, J., Carnell, S., Haworth, C. M., & Plomin, R. (2008). Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *The American Journal of Clinical Nutrition, 87*(2), 398-404.

Weedon, M. N., Lango, H., Lindgren, C. M., Wallace, C., Evans, D. M., Mangino, M., . . . Frayling, T. M. (2008). Genome-wide association analysis identifies 20 loci that influence adult height. *Nature Genetics, 40*(5), 575-583.

Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D., & Dietz, W. H. (1997). Predicting Obesity in Young Adulthood from Childhood and Parental Obesity. *New England Journal of Medicine, 337*(13), 869-873.

WHO. (2000). Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organization Technical Report Series, 894*, i-xii, 1-253.